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EXAMINER	
AEDER, SEAN E	

ART UNIT	PAPER NUMBER
1642	

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Please find below and/or attached an Office communication concerning this application or proceeding.

The time period for reply, if any, is set in the attached communication.

Office Action Summary	Application No.	Applicant(s)	
	10/773,440	FRADET ET AL.	
	Examiner	Art Unit	
	Sean E. Aeder	1642	

-- The MAILING DATE of this communication appears on the cover sheet with the correspondence address --

Period for Reply

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE 3 MONTH(S) OR THIRTY (30) DAYS, WHICHEVER IS LONGER, FROM THE MAILING DATE OF THIS COMMUNICATION.

- Extensions of time may be available under the provisions of 37 CFR 1.136(a). In no event, however, may a reply be timely filed after SIX (6) MONTHS from the mailing date of this communication.
- If NO period for reply is specified above, the maximum statutory period will apply and will expire SIX (6) MONTHS from the mailing date of this communication.
- Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133). Any reply received by the Office later than three months after the mailing date of this communication, even if timely filed, may reduce any earned patent term adjustment. See 37 CFR 1.704(b).

Status

1)☒ Responsive to communication(s) filed on *20 September 2007*.

2a)☒ This action is **FINAL**. 2b)☐ This action is non-final.

3)☐ Since this application is in condition for allowance except for formal matters, prosecution as to the merits is closed in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11, 453 O.G. 213.

Disposition of Claims

4)☒ Claim(s) *41-69* is/are pending in the application.

4a) Of the above claim(s) _____ is/are withdrawn from consideration.

5)☐ Claim(s) _____ is/are allowed.

6)☒ Claim(s) *41-69* is/are rejected.

7)☐ Claim(s) _____ is/are objected to.

8)☐ Claim(s) _____ are subject to restriction and/or election requirement.

Application Papers

9)☐ The specification is objected to by the Examiner.

10)☐ The drawing(s) filed on _____ is/are: a)☐ accepted or b)☐ objected to by the Examiner.

Applicant may not request that any objection to the drawing(s) be held in abeyance. See 37 CFR 1.85(a).

Replacement drawing sheet(s) including the correction is required if the drawing(s) is objected to. See 37 CFR 1.121(d).

11)☐ The oath or declaration is objected to by the Examiner. Note the attached Office Action or form PTO-152.

Priority under 35 U.S.C. § 119

12)☐ Acknowledgment is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d) or (f).

a)☐ All b)☐ Some * c)☐ None of:

1.☐ Certified copies of the priority documents have been received.

2.☐ Certified copies of the priority documents have been received in Application No. _____.

3.☐ Copies of the certified copies of the priority documents have been received in this National Stage application from the International Bureau (PCT Rule 17.2(a)).

* See the attached detailed Office action for a list of the certified copies not received.

Attachment(s)

1) ☒ Notice of References Cited (PTO-892)
2) ☐ Notice of Draftsperson's Patent Drawing Review (PTO-948)
3) ☒ Information Disclosure Statement(s) (PTO/SB/08) Paper No(s)/Mail Date *6/1/07; 9/20/07*.
4) ☐ Interview Summary (PTO-413) Paper No(s)/Mail Date. _____ .
5) ☐ Notice of Informal Patent Application
6) ☐ Other: _____.

Detailed Action

The Amendments and Remarks filed 9/20/07 in response to the Office Action of 3/20/07 are acknowledged and have been entered.

Claims 41-69 are pending.

Claims 41, 44-46, 50, 52, 55, 56, 63, 65, and 67 have been amended by Applicant.

Claims 41-69 are currently under examination.

Objections Withdrawn

The objection to claim 56 is withdrawn in view of amendments.

Rejections Withdrawn

The rejections under 35 U.S.C. 112, second paragraph, are withdrawn.

The rejections under 35 U.S.C. 112 first paragraph, for failing to comply with the written description requirement, are withdrawn.

The rejection of claims 41-69 under 35 U.S.C. 112, first paragraph, for failing to comply with the enablement requirement, is withdrawn.

Response to Arguments

Claim Rejections - 35 USC § 103

The following is a quotation of 35 U.S.C. 103(a) which forms the basis for all obviousness rejections set forth in this Office action:

> (a) A patent may not be obtained though the invention is not identically disclosed or described as set forth in section 102 of this title, if the differences between the subject matter sought to be patented and the prior art are such that the subject matter as a whole would have been obvious at the time the invention was made to a person having ordinary skill in the art to which said subject matter pertains. Patentability shall not be negatived by the manner in which the invention was made.

The rejection of claims 41-50, 57, 58, 61-63, and 65-68 are under 35 U.S.C. 103(a), as being unpatentable over Bussemakers et al (US 7,008,765 B1; filed 4/9/98) in view of Clements et al (The Journal of Urology, 4/99, 161: 1337-1343), is maintained for the reasons stated in the Office Action of 3/20/07 and for the reasons set-forth below.

The Office Action of 3/20/07 contains the following text:

"Bussemakers et al teaches a sequence corresponding to PCA3, SEQ ID NO:6, that is 99.5% homologous to instant SEQ ID NO:9 and shares 99.6% local similarity to the first 2036 amino acids of instant SEQ ID NO:9 (see sequence comparisons). SEQ ID NO:6 is 100% identical to instant SEQ ID NO:10 (see sequence comparisons). SEQ ID NO:6 is 89.1% homologous to instant SEQ ID NO:13 and shares 99.6% local similarity to the first 3582 polynucleotides of instant SEQ ID NO:13 (see sequence comparisons). Due to the high degree of homology between SEQ ID NO:6 and instant SEQ ID Nod 9, 10, and 13, one of skill in the art would recognize that complements of SEQ ID NO:6 would hybridize to instant SEQ ID NOs 9, 10, and 13. Bussemakers et al further teaches a method for determining a predisposition for or the presence of prostate cancer in a patient comprising (a) performing an RT-PCR RNA amplification assay on a prostate biopsy sample comprising at least one prostate cell of said patient, or nucleic acid thereof, using a first primer pair specific to SEQ ID NO:6, (b) performing a second RT-PCR RNA amplification assay on said sample using a second primer pair specific to PSA, (c) detecting in said sample an amount of PCA3 and PSA mRNA; and (d) wherein an increased level of PCA3 mRNA, as compared to the level of PCA3 mRNA in a urine sample from a normal subject, indicates that said patient will develop prostate cancer or that said patient has prostate cancer; and (e) wherein an absence of PCA3 mRNA or a lower level of PCA3 mRNA, as compared to the level of PCA3 mRNA

in a sample from a normal subject, indicates that said patient will not develop prostate cancer and that said patient does not prostate cancer, when PSA mRNA is detected (Example 2, in particular). Further, without claiming any stringency of hybridization, PSA would hybridize to kallikrein 2. Bussemakers further teaches a method wherein said amplification assay is TMA (column 37, in particular). Bussemakers further teaches a method wherein said amplification of PCA3 and said PSA mRNA is performed simultaneously (column 36, in particular). Bussemakers further teaches a method wherein said detection is performed by chemiluminescence (paragraph bridging columns 15-16, in particular). By teaching PSA mRNA is detected in both benign and malignant prostate samples and that the level of PSA mRNA is not a reliable indicator of prostate cancer disease state, Bussemakers teaches a method wherein detection of PSA would validate a negative result for PCA3 detection in that the detection of PSA mRNA is indicative of the presence of prostate cells in a sample that did not display elevated PCA3 levels (Example 2, in particular). Bussemakers further teaches a method wherein RNA is extracted using a target capture method (Example 2, in particular). Bussemakers further teaches a method wherein said detection of PCA3 is carried out using chemiluminescent labels in a homogenous detection method (see paragraph bridging pages 15-16 and Example 2, in particular). Bussemakers further teaches a method wherein said amplification of PCA3 and said second prostate specific nucleic acid is performed simultaneously in one container (see column 36, in particular).

Bussemakers does not specifically teach methods using a urine sample, a voided urine sample from a patient having an increased number of prostate cells therein, a urine sample containing semen, or a urine sample collected following a digital rectal exam. However, these deficiencies are made up in the teachings of Clements et al.

Clements et al teaches a method of using RT-PCR to detect PSA mRNA as a selective marker of prostate cells in urine from normal and prostate cancer patients (see abstract, in particular). Clements et al further teaches methods of using RT-PCR to detect prostate cells in urine containing semen, which was collected as the first urinary void immediately after masturbation, from normal and prostate cancer patients (left column of page 1338, in particular). As compared to the female urine samples obtained by Clements et al, male urine samples had an increased number of prostate cells (left column of page 1339, in particular). Clements et al further teaches methods using urine samples from patients that have had digital rectal exams (left column of page 1338, in particular). Clements et al further teaches that PSA mRNA expression is intended to be used as a marker for prostate cells when screening patients for mRNA markers that are upregulated in prostate cancer cells of patients with prostate cancer (see paragraph flanking pages 1340-1341, in particular).

One of ordinary skill in the art at the time the invention was made would have been motivated to use male urine containing semen that was collected following a digital rectal exam, as taught by Clemens et al, as the sample of the method taught by Bussemakers et al because one of skill in the art would recognize that a digital rectal exam would be used as a quick method to screen for prostate cancer and obtaining male urine containing semen that was collected following said digital rectal exam is less unpleasant and invasive than collecting a prostate biopsy (left column of page 1337 of

Clements et al, in particular). In addition to reasons obvious from the teachings of Bussemakers et al, one of skill would have been further motivated to use PSA mRNA as a marker for prostate cells when screening for PCA3 mRNA in urine containing semen that was collected following a digital rectal exam because Clemens et al teaches that PSA mRNA expression is intended to be used as a marker for prostate cells when screening patients for mRNA markers that are upregulated in patients with prostate cancer (see paragraph flanking pages 1340-1341, in particular) and Bussemakers et al teaches PCA3 demonstrates 20-fold overexpression in prostate cancer cells as compared to PCA3 expression levels in prostate cells from patients without prostate cancer (column 37 of Bussemakers et al, in particular). One of ordinary skill in the art at the time the invention was made would have had a reasonable expectation of success for using male urine containing semen that was collected following a digital rectal in detecting PCA3 mRNA and PSA in the method taught by Bussemakers et al because Clements et al teaches a method of detecting mRNA expressed by normal prostate cells and prostate cancer cells in male urine containing semen that was collected following a digital rectal exam (pages 1338-1339, in particular). Therefore, the invention as a whole would have been prima facie obvious to one of ordinary skill in the art at the time the invention was made, absent unexpected results."

The Reply of 9/20/07, Applicant disagrees with Examiner's statement that Bussemakers teaches "wherein an absence of PCA3 mRNA or a lower level of PCA3 mRNA as compared to the level of PCA3 mRNA in a sample from a normal subject, indicates that said patient will not develop prostate cancer and that said patient does not have prostate cancer, when PSA mRNA is detected" and states that Bussemakers is silent on detection of an additional prostate-specific marker from the same sample to validate results using PCA3. Applicant further disagrees with Examiner's statement that Bussemakers teaches a method in which the "amplification of PCA3 and said PSA and mRNA is performed simultaneously". Applicant further states that Bussemakers indicates (lines 6-8 of column 37 and lines 28-37 of column 38) that PSA expression was decreased in malignant versus benign tissues, which Applicant interprets to indicate that PSA may not be useful to quantify normal and cancer cells because

expression of the gene product is not constant. Applicant states that the teaching of Bussemakers would provide reasons not to use nucleic acid markers such as PSA, human kallikrein 2, PSMA..., as markers to validate the diagnostic methods of the invention. Applicant further argues that the use of urine which does not contain semen is not validated, but rather taught away, by Clements and Clements does not teach that a regular urine sample (e.g., a voided urine sample pre-ejaculation) is a suitable sample for the detection of PSA. Applicant further argues that the samples of Clements were not obtained following digital rectal examinations (DRE) and DRE only served to recruit patient in the study. Applicant further disagrees with Examiner's statement that Clements "teaches that the PSA mRNA expression is intended to be used as a marker for prostate cells when screening patients for mRNA markers that are up regulated in prostate cancer cells of patients with prostate cancer". Applicant further indicates that while Clements teaches a protein marker (Apo-D) is upregulated in urine from prostate cancer patients and PSA mRNA levels in urine do not differentiate between urine from prostate cancer patients and normal patients, Clements does not teach that RT-PCR from urine samples *per se* can be used to detect prostate cells therein and fails to teach a usefulness in using mRNA detection for assessing the malignant state of the prostate. Applicant further cites Fradet et al (Urology, 2004, 64(2):311-316) and argues that a "reasonable likelihood" of success that a urine-based test could be used to diagnose prostate cancer based on the detection of PCA3 RNA and a prostate specific RNA would not be predicted from the prior art. Applicant concludes that Bussemakers and Clements (together or any other reference cited in the Office Action of 3/20/07) do not

teach or suggest a method combining the detection of both PCA3 mRNA and a prostate-specific mRNA in a urine sample having not been obtained immediately following ejaculation, to determine the presence / absence / predisposition to develop prostate cancer when in the absence of detection of PAC3 the prostate specific marker is detected and it does not teach or suggest every element of independent claims 41 and 67.

The arguments found in the Reply of 9/20/07 have been carefully considered, but are not deemed persuasive. In regards to the statement that Bussemakers does not teach a method "wherein an absence of PCA3 mRNA or a lower level of PCA3 mRNA as compared to the level of PCA3 mRNA in a sample from a normal subject, indicates that said patient will not develop prostate cancer and that said patient does not have prostate cancer, when PSA mRNA is detected", Bussemakers clearly teaches that PSA is *exclusively* expressed in cells of the prostate (lines 25-28 of column 2, in particular) and that an absence of PCA3 mRNA or a lower level of PCA3 mRNA in a sample comprising prostate cells from a patient as compared to the level of PCA3 mRNA in a sample comprising prostate cells from a normal subject, indicates that said patient will not develop prostate cancer and that said patient does not have prostate cancer (see Example 2, in particular). Therefore, one of ordinary skill in the art at the time the invention was made would have been motivated to detect PSA mRNA in a sample when detecting PCA3 mRNA in order to verify that said sample comprises prostate cells because PSA is exclusively expressed in prostate cells and differential expression of PCA3 mRNA in prostate cells indicates whether or not a patient has or will develop

prostate cancer. Further, Clemens et al teaches that PSA mRNA expression is intended to be used as a marker for prostate cells when screening patients for markers that are upregulated in patients with prostate cancer (see paragraph flanking pages 1340-1341, in particular) and Bussemakers et al teaches PCA3 demonstrates 20-fold overexpression in prostate cancer cells as compared to PCA3 expression levels in prostate cells from patients without prostate cancer (column 37 of Bussemakers et al, in particular).

In regards to the argument that Bussemakers does not teach a method in which the "amplification of PCA3 and said PSA and mRNA is performed simultaneously", Bussemakers teaches (at lines 57-63 of column 36) amplification of PCA3 and PSA and mRNA performed in a single semi-quantitative RT-PCR analysis. Further, claims drawn to methods of "simultaneously" amplifying two particular markers encompass methods of detecting said two particular makers during said method (see instant claim 50). As discussed in the paragraph above, amplification of PCA3 and said PSA and mRNA in the same method would be performed in the combined teachings of Bussemakers and Clements. Therefore, the combined teachings of Bussemakers and Clements teach a method whereby amplification of PCA3 and said PSA and mRNA is performed simultaneously.

In regards to the statements that (1) Bussemakers indicates (lines 6-8 of column 37 and lines 28-37 of column 38) that PSA expression was decreased in malignant versus benign tissues, which Applicant interprets to indicate that PSA may not be useful to quantify normal and cancer cells because expression of the gene product is not

constant, and (2) the teaching of Bussemakers would provide reasons not to use nucleic acid markers such as PSA, human kallikrein 2, PSMA..., as markers to validate the diagnostic methods of the invention, the art teaches that PSA expression is used to identify prostate cells and that the marker taught by Bussemakers (PCA3) is used in methods of differentiating normal prostate cells from cancerous/pre-cancerous prostate cells. As stated above, Bussemakers clearly teaches that PSA is *exclusively* expressed in cells of the prostate (lines 25-28 of column 2, in particular) and that an absence of PCA3 mRNA or a lower level of PCA3 mRNA in a sample comprising prostate cells from a patient as compared to the level of PCA3 mRNA in a sample comprising prostate cells from a normal subject, indicates that said patient will not develop prostate cancer and that said patient does not have prostate cancer (see Example 2, in particular). Therefore, one of ordinary skill in the art at the time the invention was made would have been motivated to detect PSA mRNA in a sample when detecting PCA3 mRNA in order to verify that said sample comprises prostate cells because PSA is exclusively expressed in prostate cells and differential expression of PCA3 mRNA in prostate cells indicates whether or not a patient has or will develop prostate cancer. Further, Clemens et al teaches that PSA mRNA expression is intended to be used as a marker for prostate cells when screening patients for markers that are upregulated in patients with prostate cancer (see paragraph flanking pages 1340-1341, in particular) and Bussemakers et al teaches PCA3 demonstrates 20-fold overexpression in prostate cancer cells as compared to PCA3 expression levels in prostate cells from patients without prostate cancer (column 37 of Bussemakers et al, in particular).

In regards to the argument that the use of urine which does not contain semen is not validated, but rather taught away, by Clements and Clements does not teach that a regular urine sample (e.g., a voided urine sample pre-ejaculation) is a suitable sample for the detection of PSA, Applicant is arguing limitations not commensurate in scope with the claims. The pending claims do not recite methods where urine "does not contain semen". Rather, the pending claims recite methods wherein the urine sample is from a patient "having not been obtained immediately following ejaculation". Clearly, one of skill in the art would recognize that urine is obtained immediately following urination (and not ejaculation).

In regards to the argument that the samples of Clements were not obtained following digital rectal examinations (DRE) and DRE only served to recruit patient in the study, the combined teaching of Bussemakers and Clements teach methods of detecting prostate cancer. Clearly, as taught by Clements (left column of page 1338, in particular), one of skill in the art would recognize that digital rectal examinations are performed in order to detect prostate cancer and that combining various detection methods (digital rectal examination and detection of makers of prostate cancer) is obvious in order to accurately determine the status of a particular patient.

In regards to the argument that Clements does not teach "that the PSA mRNA expression is intended to be used as a marker for prostate cells when screening patients for mRNA markers that are up regulated in prostate cancer cells of patients with prostate cancer", Clements teaches PSA RT-PCR is a reliable indicator of prostate cells in urine (see abstract, in particular) and Clements et al further teaches a method

wherein other potential mRNA and potential protein markers were used to determine whether said potential markers are prostate cancer specific. Because Bussemakers et al teaches PCA3 demonstrates 20-fold overexpression in prostate cancer cells as compared to PCA3 expression levels in prostate cells from patients without prostate cancer (column 37 of Bussemakers et al, in particular), one of skill in the art would use PSA mRNA expression to determine whether or not prostate cells are present in samples when using PCA3 expression levels as a marker for prostate cancer.

In regards to the argument that while Clements teaches a protein marker (Apo-D) is upregulated in urine from prostate cancer patients and PSA mRNA levels in urine do not differentiate between urine from prostate cancer patients and normal patients, Clements does not teach that RT-PCR from urine samples *per se* can be used to detect prostate cells therein and fails to teach a usefulness in using mRNA detection for assessing the malignant state of the prostate, use of mRNA from urine samples and RT-PCR to detect prostate cells and for assessing the malignant state of the prostate is taught by the combined teachings of Bussemakers and Clements for the reasons stated above.

In regards to the argument that a "reasonable likelihood" of success that a urine-based test could be used to diagnose prostate cancer based on the detection of PCA3 RNA and a prostate specific RNA would not be predicted from the prior art, the Examiner disagrees. The prior art clearly teaches PCA3 mRNA is differentially expressed in prostate cancer cells found in urine of patients with prostate cancer, as compared to prostate cells found in urine of normal subjects (see Example 2 of

Bussemakers, in particular). Further, the prior at teaches that PSA is a well-known marker for prostate cells (see lines 25-28 of column 2 in Bussemakers and page 1341 of Clements, in particular). Therefore, there is a "reasonable likelihood" of success that a urine-based test would diagnose prostate cancer based on the detection of PCA3 mRNA in samples comprising PSA mRNA because samples comprising PSA mRNA comprise prostate cells and differential expression of PCA3 mRNA in prostate cells would lead one to a prostate cancer diagnosis.

Claim Rejections - 35 USC § 103

The rejection of claims 41-50, 57, 58, 61-68 under 35 U.S.C. 103(a), as being unpatentable over Bussemakers et al (US 7,008,765 B1; filed 4/9/98) in view of Clements et al (The Journal of Urology, 4/99, 161: 1337-1343), in further view of Cheung et al (Journal of Clinical Microbiology, 10/94, 2593-2597), is maintained for the reasons stated in the Office Action of 3/20/07 and for the reasons set-forth below.

In the Reply of 9/20/07, Applicant argues that the teachings of Cheung do not correct alleged defects of the combination of Bussemakers and Clements.

The amendments to the claims and the arguments found in the Reply of 9/20/07 have been carefully considered, but are not deemed persuasive. Arguments addressing alleged defects of Bussemakers and Clements are found above.

Claim Rejections - 35 USC § 103

The rejection of claims 41-50, 57, 58, 59-63, and 65-69 under 35 U.S.C. 103(a), as being unpatentable over Bussemakers et al (US 7,008,765 B1; filed 4/9/98) in view of Clements et al (The Journal of Urology, 4/99, 161: 1337-1343), and in further view of Baret (EP 0 256 932 A2; 2/24/88), is maintained for the reasons stated in the Office Action of 3/20/07 and for the reasons set-forth below.

In the Reply of 9/20/07, Applicant argues that the teachings of Baret do not correct alleged defects of the combination of Bussemakers and Clements.

The amendments to the claims and the arguments found in the Reply of 9/20/07 have been carefully considered, but are not deemed persuasive. Arguments addressing alleged defects of Bussemakers and Clements are found above.

Claim Rejections - 35 USC § 103

The rejection of claims 41-51, 54, 57, 58, 61-63, and 65-68 under 35 U.S.C. 103(a), as being unpatentable over Bussemakers et al (US 7,008,765 B1; filed 4/9/98) in view of Clements et al (The Journal of Urology, 4/99, 161: 1337-1343), in further view of Buck et al (1999, Biotechniques, 27(3):528-536), is maintained for the reasons stated in the Office Action of 3/20/07 and for the reasons set-forth below.

In the Reply of 9/20/07, Applicant argues that the teachings of Buck do not correct alleged defects of the combination of Bussemakers and Clements.

The amendments to the claims and the arguments found in the Reply of 9/20/07 have been carefully considered, but are not deemed persuasive. Arguments addressing alleged defects of Bussemakers and Clements are found above.

Claim Rejections - 35 USC § 103

The rejection of claims 41-50, 52, 53, 55, 56, 57, 58, 61-63, and 65-68 under 35 U.S.C. 103(a), as being unpatentable over Bussemakers et al (US 7,008,765 B1; filed 4/9/98) in view of Clements et al (The Journal of Urology, 4/99, 161: 1337-1343), in further view of Schlegel et al (US 2002/0168638 A1; filed 1/24/01), is maintained for the reasons stated in the Office Action of 3/20/07 and for the reasons set-forth below.

In the Reply of 9/20/07, Applicant argues that the teachings of Schlegel do not correct alleged defects of the combination of Bussemakers and Clements.

The amendments to the claims and the arguments found in the Reply of 9/20/07 have been carefully considered, but are not deemed persuasive. Arguments addressing alleged defects of Bussemakers and Clements are found above.

Summary

No claim is allowed.

Conclusion

THIS ACTION IS MADE FINAL. Applicant is reminded of the extension of time policy as set forth in 37 C.F.R. ' 1.136(a). A shortened statutory period for response to

this Final Action is set to expire three months from the date of this action. In the event a first response is filed within two months of the mailing date of this Final Action and the advisory action is not mailed until after the end of the three-month shortened statutory period, then the shortened statutory period will expire on the date the advisory action is mailed, and any extension fee pursuant to 37 C.F.R. '1.136(a) will be calculated from the mailing date of the advisory action. In no event will the statutory period for response expire later than six months from the date of this Final Action.

Any inquiry concerning this communication or earlier communications from the examiner should be directed to Sean E. Aeder, Ph.D. whose telephone number is 571-272-8787. The examiner can normally be reached on M-F: 8:30-5:00.

If attempts to reach the examiner by telephone are unsuccessful, the examiner's supervisor, Shanon Foley can be reached on 571-272-0898. The fax phone number for the organization where this application or proceeding is assigned is 571-273-8300.

Information regarding the status of an application may be obtained from the Patent Application Information Retrieval (PAIR) system. Status information for published applications may be obtained from either Private PAIR or Public PAIR. Status information for unpublished applications is available through Private PAIR only. For more information about the PAIR system, see http://pair-direct.uspto.gov. Should you have questions on access to the Private PAIR system, contact the Electronic Business Center (EBC) at 866-217-9197 (toll-free).

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